09/932,678

WEST Search History

DATE: Monday, August 05, 2002

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT; PLUR=YES; OP=OR			
L4	RRN3	2	L4
DB=PGPB,JPAB,EPAB; PLUR=YES; OP=OR			
L3	RRN3	1	L3
L2	L1	0	L2
DB=USPT; PLUR=YES; OP=OR			
L1	rrn3	2	L1

END OF SEARCH HISTORY

Searches for 09/932,678 RPL and Pratt (West)

Your SELECT statement is:
s rrn3 and ribosom?

Items	File
8	5: Biosis Previews(R)_1969-2002/Jul W4
4	34: SciSearch(R) Cited Ref Sci_1990-2002/Aug W1
4	35: Dissertation Abs Online_1861-2002/Jul
3	71: ELSEVIER BIOBASE_1994-2002/Aug W1
5	73: EMBASE_1974-2002/Jul W4
1	144: Pascal_1973-2002/Aug W1
11	155: MEDLINE(R)_1966-2002/Jul W4
1	159: Cancerlit_1975-2002/Jun
1	399: CA SEARCH(R)_1967-2002/UD=13706

9 files have one or more items; file list includes 28 files.

?b 5, 155, 159
05aug02 08:48:41 User264783 Session D159.2
$0.64 0.366 DialUnits File411
$0.64 Estimated cost File411
$0.43 TELNET
$1.07 Estimated cost this search
$1.19 Estimated total session cost 0.517 DialUnits

SYSTEM:OS - DIALOG OneSearch
File 5:Biosis Previews(R) 1969-2002/Jul W4
(c) 2002 BIOSIS
***File 5: Alert feature enhanced for multiple files, duplicates**
removal, customized scheduling. See HELP ALERT.
File 155:MEDLINE(R) 1966-2002/Jul W4
***File 155: Alert feature enhanced for multiple files, duplicates**
removal, customized scheduling. See HELP ALERT.
File 159:Cancerlit 1975-2002/Jun
(c) format only 2002 Dialog Corporation
***File 159: The file has been reloaded. Accession Numbers have changed.**

Set	Items	Description
S1	20	RRN3 AND RIBOSOM?
S2	14	RD (unique items)

2/9/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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13693732 BIOSIS NO.: 200200322553
Rrn3 **phosphorylation, cycloheximide and rDNA transcription: Cycloheximide inhibits the phosphorylation of** Rrn3 **and the interaction between** Rrn3 **and rpa43.**
AUTHOR: Rothblum Lawrence(a); Hirschler-Laszkiewicz Iwona(a); Hu Qiyue(a); Cavanaugh Alice(a)
AUTHOR ADDRESS: (a)Geisinger Clinic, Weis Center for Research, 100 N. Academy Avenue, Danville, PA, 17822**USA
JOURNAL: FASEB Journal 16 (4):pA1 March 20, 2002
MEDIUM: print
CONFERENCE/MEETING: Annual Meeting of the Professional Research Scientists on Experimental Biology New Orleans, Louisiana, USA April 20-24, 2002
ISSN: 0892-6638
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Yu and Feigelson reported that cycloheximide (CHX) caused a rapid cessation of nucleolar RNA synthesis. Subsequently, TIF-IA and Factor C* were identified as factors that could complement extracts of serum starved cells, and TFIC was identified as that required to complement extracts of P1798 cells treated with dexamethasone. However, it was not clear if these were the same or different proteins. TIF-IA is the mouse homologue of yeast **Rrn3** . Mammalian **Rrn3** can complement extracts from CHX treated cells. However, the mechanism by which **Rrn3** activity is inactivated by CHX is not known. We found that **Rrn3** can complement

extracts of cells treated with CHX, but not extracts of DEX treated lymphoma cells, demonstrating that **Rrn3** /TIF-IA and TFIC are not the same activities. We have confirmed that **Rrn3** can interact with both RPI and SL1. We found that CHX inhibits **Rrn3** phosphorylation and causes the dissociation of **Rrn3** from RPI. Moreover, treatment with CHX results in the inhibition of the formation of a **Rrn3** -rpa43 complex in vivo. Subsequent experiments demonstrated that **Rrn3** could interact with rpa43 in vitro, and that dephosphorylated **Rrn3** could neither interact with rpa43 nor reconstitute transcription.

2/9/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13484528 BIOSIS NO.: 200200113349
Differential roles of phosphorylation in the formation of transcriptional active RNA polymerase I.
AUTHOR: Fath Stephan; Milkereit Philipp; Peyroche Gerald; Riva Michel; Carles Christophe; Tschochner Herbert(a)
AUTHOR ADDRESS: (a)Institute fuer Biochemie, Universitaet Heidelberg, Im Neuenheimer Feld 328, D-69120, Heidelberg**Germany E-Mail: im4@popix.urz.uni-heidelberg.de
JOURNAL: Proceedings of the National Academy of Sciences of the United States of America 98 (25):p14334-14339 December 4, 2001
MEDIUM: print
ISSN: 0027-8424
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Regulation of rDNA transcription depends on the formation and dissociation of a functional complex between RNA polymerase I (pol I) and transcription initiation factor Rrn3p. We analyzed whether phosphorylation is involved in this molecular switch. Rrn3p is a phosphoprotein that is predominantly phosphorylated in vivo when it is not bound to pol I. In vitro, Rrn3p is able both to associate with pol I and to enter the transcription cycle in its nonphosphorylated form. By contrast, phosphorylation of pol I is required to form a stable pol I-Rrn3p complex for efficient transcription initiation. Furthermore, association of pol I with Rrn3p correlates with a change in the phosphorylation state of pol I in vivo. We suggest that phosphorylation at specific sites of pol I is a prerequisite for proper transcription initiation and that phosphorylation/dephosphorylation of pol I is one possibility to modulate cellular rDNA transcription activity.

2/9/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13038370 BIOSIS NO.: 200100245519
hRRN3 is essential in the SL1-mediated recruitment of RNA polymerase I to rRNA gene promoters.
AUTHOR: Miller Gail; Panov Kostya I; Friedrich J Karsten; Trinkle-Mulcahy Laura; Lamond Angus I; Zomerdijk Joost C B M(a)
AUTHOR ADDRESS: (a)Division of Gene Regulation and Expression, School of Life Sciences, Wellcome Trust Biocentre, University of Dundee, Dundee, DD1 5EH: j.zomerdijk@dundee.ac.uk**UK
JOURNAL: EMBO (European Molecular Biology Organization) Journal 20 (6):p 1373-1382 March 15, 2001
MEDIUM: print
ISSN: 0261-4189
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: A crucial step in transcription is the recruitment of RNA polymerase to promoters. In the transcription of human rRNA genes by RNA Polymerase I (Pol I), transcription factor SL1 has a role as the essential core promoter binding factor. Little is known about the

mechanism by which Pol I is recruited. We provide evidence for an essential role for hRRN3, the human homologue of a yeast Pol I transcription factor, in this process. We find that whereas the bulk of human Pol I complexes (Ialpha) are transcriptionally inactive, hRRN3 defines a distinct subpopulation of Pol I complexes (Ibeta) that supports specific initiation of transcription. Human **RRN3** interacts directly with TAFI110 and TAFI63 of promoter-selectivity factor SL1. Blocking this connection prevents recruitment of Pol I beta to the rDNA promoter. Furthermore, hRRN3 can be found in transcriptionally autonomous Pol I holoenzyme complexes. We conclude that hRRN3 functions to recruit initiation-competent Pol I to rRNA gene promoters. The essential role for hRRN3 in linking Pol I to SL1 suggests a mechanism for growth control of Pol I transcription.